Radiation Protection: Exposures in Low-Earth Orbit

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The guidelines that have been the basis of NASA's radiation exposure limits since 1989 have been re-examined and updated in NCRP Report 132 (NCRP, 2000). New career dose limits for stochastic effects have been recommended, which are lower than the current limits but continue to be based on three per cent excess mortality and take into account age at first exposure and gender. The limits range from 0.4 Sv for a female first exposed at 25 years of age to 3.0 Sv for a male first exposed at 55 years of age. The career dose limits have been reduced because of new risk estimates that come mainly from the studies of the atomic bomb survivors. The risk estimates will change as more data becomes available but should remain relatively stable for some years. Separate career limits are set for deterministic effects and limits for shorter periods, such as one year are also recommended. These limits, which have in the past been given in sieverts are now expressed in gray equivalents (GY-Eq) and are obtained by multiplying the organ doses in Gy by the appropriate RBE. It is recommended that the RBEs be determined from the threshold dose for the specific effect.

The appropriateness of the career limits depends on: 1) dosimetry, in particular the relative contribution of radiations of different qualities, 2) the accuracy of the risk estimates, which depends greatly on how to estimate the risk for exposures at very low dose rates from data obtained from a population exposed at a high dose rate, 3) the acceptability of basing the limits on a 3% excess mortality; the maximum current radiation protection limits for occupationally exposed populations on earth could theoretically result in a 3% excess mortality and 4) the precision of the values of RBEs for threshold doses of deterministic effects: many of the values have to be extrapolated from data obtained in animal studies.

The risk estimates for both stochastic and deterministic effects will be improved when:1) the effects of neutrons of energies greater than 2 Mev are known more accurately, 2) the effects of protraction of proton irradiation are assessed and 3) the threshold doses for deterministic effects from exposures at low dose rates to protons, neutrons and heavy-charged particles are known more precisely.

In the case of the assessment of the risks of exposures in deep space the same information is required with the addition of a much better understanding of the potential risk to the CNS from protracted exposures to heavy ions and the possibility of acute tissue damage from very large solar particle events. Neither of these aspects will be solved by cellular or molecular studies and require well-conceived animal experiments and whatever can be gleaned from patients treated with proteins and heavy ions.

CNS and Radiation

The central nervous system (CNS) is considered a relatively radio-resistant organ; it consists of neurons differing markedly in size and their number per unit area. There are several nuclei or centers that consist of closely packed neuron cell bodies, for example, the respiratory and cardiac centers in the floor of the fourth ventricle. In the cerebral cortex the large neuron cell bodies, such as Betz cells, are separated by a considerable distance. The other tissues of importance are the neuroglia, which are the supporting cells and consist of astrocytes, oligodendroglia, and microglia. These cells permeate and support the nervous tissue of the CNS, binding it together like a scaffold that also supports the vasculature. The most numerous of the neuroglia is type I astrocytes, which make up about half the brain greatly outnumbering the neurons. Neuroglia retain the capability of cell division in contrast to neurons, and therefore the responses to irradiation differ between the cell types. The third type of tissue is the vasculature which exhibits a comparable vulnerability for radiation damage to that found elsewhere in the body (Rheinhold, 1980). Oligodendrocytes and the endothelial cells of the microvasculature appear to be the targets for radiation-induced damage that account for major aspects of the pathogenesis of brain damage that can occur after high doses of low-LET radiation.

The radiation effects, both stochastic and deterministic in nature, can occur in the CNS. Tumors may arise from the neuroglia, such as glioblastomas, and from the connective tissue, called the meninges, that encapsulates the brain and spinal cord. There is a marked age dependency in the susceptibility for the induction of tumors of the CNS by radiation, and the risk in adults is small. The major concern in relation to space radiation, and in particular HZE particles, are deterministic effects. The concern about HZE particles is that the pattern of energy deposition is so different from that of other radiation qualities for which we have some understanding of the radiobiological effects. In the case of HZE particles, especially the higher Z particles, the particle can traverse a large number of contiguous or neighboring cells with a track of dense ionizations. The impact of such particles is compounded by the energy deposited by the delta rays that extend from the main particle track to reach cells lateral to the track. Considering this distribution of energy it is not surprising that there is little or no confidence in estimating the risk of damage to CNS based on data obtained with low-LET radiation.

It was the distinct pattern of potential damage that raised concerns many years ago that was expressed in terms of microlesions. The concern today in estimating the risk of significant damage to the CNS that might result from long duration missions in deep space is the simple fact that we do not know whether or not there is a risk of clinically important damage, and if so, what is the probability and potential severity of such damage. The risk of stochastic effects may not be known with sufficient accuracy, but in the case of late deterministic effects on the CNS we just do not know even if there is a risk. However, there is sufficient experimental data to reinforce the concerns and to realize the urgent need for further study.

The effects of high doses of low-LET radiation are known reasonably well. The tolerance dose for the brain for fractionated exposures is in terms of thousands of grays (Kramer et al., 1972). Cellular changes, degeneration, and necrosis can occur.

It has been estimated that about 1 particle of a Z between 3 and 28 will traverse every nucleus per year (Curtis et al., 1995; see also Curtis et al., in press). HZE particles are more effective and the effects appear earlier than after low-LET radiation (Lett, 1980). Damage in the forebrain of rabbits has been reported after exposure to 0.5 G of Ne ions. It is therefore essential to know what the effects, especially late effects, of such a bombardment may be. Unfortunately there are few, quantitative data for the loss of cells due to heavy ion irradiation. Madi and Gupta et al. have indicated marked losses of tyrosine hydroxylase immunoreactivity in the substantia nigra after exposure to Fe ions. These results have been interpreted to indicate cell loss, however the precise relationship between the observed changes and permanent loss of cells is not clear.

An important question is whether neurons traversed by HZE particles and which survive develop changes as HZE particles and which survive develop changes as a late consequence of the damage they incurred. This question has been addressed using retinal photoreceptors, rods as a surrogate for neurons in the CNS. Lett and his coworkers (1987, 1994, 1996) found changes in the DNA of these cells with time after irradiation. After exposure to low-LET radiation or HZE particles, the initial radiation-induced damage was repaired, but a subsequent breakdown of DNA occurred with age. Exposure to. HZE particles resulted in the secondary changes occurring at a younger age than after exposure to low-LET radiation. Loss of rods from rabbit retinas occurred with age but more markedly after exposure to irradiation, especially Fe ions. Complete dose-response data are not yet available, thus it is not known whether the fluences that would be experienced during missions in deep space pose a significant risk.

It has been known for some time that changes in behavior of rodents could be detected after low doses of heavy ions (Hunt et al., 1989, Rabin et al., 1987, 1989, 1991, 1994). The possible underlying mechanisms to these changes are starting to be understood (Joseph et al., 1992, 1993). Joseph and his colleagues (1992, 1993) have found that low doses of Fe ions can induce changes in dopaminergic function and that this, in turn, may alter a number of dopamine-mediated behaviors, in particular certain conditioned taste aversions (CT A). The CT A test assesses the avoidance of normally acceptable food as a result of exposure to some toxic agent such as radiation (Riley and Tuck, 1985). The role of the dopaminergic system in radiation-induced changes in CT A is suggested by the fact that amphetamine-induced CT A, which depends on the dopaminergic system, is affected by radiation, whereas lithium chloride-induced CT A that does not involve the dopaminergic system is not affected by radiation. Rabin et al. (1989, 1991) have established that the degree ofCTA due to radiation is LET dependent and that Fe ions are the most effective of the various low- and high-LET radiations that have been tested. Doses of about 20 cGY of ^{56}Fe ions appear to have an effect on CTA.

Motor Function

It has been shown by Joseph et al. (1992) that low doses of Fe ions reduces performance as tested by the "wire suspension" test. This test, which assesses the time a rodent will or can hang on to a wire, is considered a measure of motor function.

In Summary

While no reliable estimate of risk of important damage to the CNS can yet be given, there are enough data to indicate much more must be known before risk estimates of the effects of exposures in deep space can be made with any confidence. The accumulated evidence from the reported studies on DNA damage, loss of neurons, altered behavior, and motor function is sufficient to require a careful assessment of the total risk to the CNS from exposure to HZE particles.

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